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LEAD POISONING AND OTHER MORTALITY FACTORS IN TRUMPETER SWANS

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Abstract – Lead poisoning and other causes of mortality of trumpeter swans were investigated. Necropsies or Pb concentrations in livers were available for 72 trumpeter swans (*Cygnus buccinator*) found dead in seven western states from 1976 to 1987; data from other published and unpublished sources (1925 to 1987) also are summarized. Ingestion of lead artifacts (shotgun pellets or fishing sinkers) accounted for about 20% of the known mortality of trumpeter swans in the tri-state area of Idaho, Montana and Wyoming, where the population has been declining for several decades. In western Washington, the incidence of lead-induced mortality was higher and accounted for nearly 50% of the known mortalities. Maximum Pb concentrations (wet weight) in the livers of birds found dead and in whole blood from captured swans were 37 and 0.71 μg/g, respectively. Other elements, including Cd, Cu and Zn, were generally not elevated in blood and tissue samples. It is not certain that lead toxicosis is related to the decline of the tri-state swan population. Other causes of mortality in swans include gunshot, disease and traumatic injuries (impact).

Keywords – Trumpeter swans Lead poisoning Lead shot Mortality Fishing sinkers

INTRODUCTION

The adverse impact of lead shot on wildlife, particularly waterfowl, is well documented [1], and serious problems still persist in many areas [2]. Swans seem particularly vulnerable to lead poisoning, and problems have been documented in several species in Europe and North America. The mute swan (*Cygnus olor*) population has declined in parts of England over the past 25 years, primarily from the ingestion of lead fishing sinkers, which causes lethal and sublethal (lowered reproductive success) effects [3–7]. Lead poisoning also has been documented in mute swans in Sweden [8], Denmark [9], Norway [10] and Scotland [11], and in whooper swans (*Cygnus cygnus*) in Norway [10], Germany [12] and Scotland [11]. In North America, lead poisoning in tundra swans (*Cygnus columbianus*) has been related to the ingestion of lead shot [1,13,14] as well as the ingestion of sediments or biota containing lead that originated from mining and smelting activities [15,16]. Trumpeter swans (*Cygnus buccinator*) also have succumbed to lead toxicosis related to ingested shot, and wintering birds in southwestern British Columbia and northwestern Washington seem particularly vulnerable to plumbism or lead poisoning [17–20].

The population of trumpeter swans that occupies parts of the tri-state area of Idaho, Montana and Wyoming has declined since 1964 [21]. This study was undertaken to determine the role of lead and other contaminants in that decline. We report levels of Pb and other elements in blood samples collected in 1984 and 1985 from trumpeter swans at Red Rock Lakes National Wildlife Refuge (RRLNWR) in Montana and Malheur National

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Wildlife Refuge (MNWR) in Oregon, and document mortality factors and concentrations of Pb and other elements in the tissues of swans found dead after 1975 in the western United States. The mortality records for trumpeter swans from other studies in the United States and Canada also are summarized.

METHODS

Most of the original data reported here were derived from trumpeter swans found dead since 1975; specimens that were in suitable condition were frozen and shipped to the National Wildlife Health Center (NWHC) in Madison, Wisconsin, for necropsy examination. Each carcass was thawed and examined for gross pathological changes. When appropriate, samples of each carcass were collected for bacteriological and virological culture. Samples taken for histologic evaluation were fixed in 10% buffered formalin, cut at 6 μ, mounted on glass slides and stained according to standard histological procedures. Signs of lead poisoning determined from gross necropsy usually included roughened, bile-stained or sloughing gizzard pads; partial or complete gizzard impaction; and detection of ingested lead fragments in the gizzard.

Other swans were examined in the field for gross lesions of lead intoxication as well as other causes of death. Tissues were removed, weighed and placed in jars (previously rinsed with acetone, hexane and dilute nitric acid); the samples were frozen until analyzed either at the NWHC or the Patuxent Wildlife Research Center (PWRC) in Laurel, Maryland.

All livers and whole-blood samples were analyzed for Pb; additional analyses were conducted on from 1 to 13 livers for Al, As, Cr, Cu, Fe, Hg, Mg, Mn, Ni, Se and Zn; 3 kidney samples for Cd; 4 femurs for Fl and Pb; and 3 to 10 blood samples for Cu, Zn, Ni, Hg, As, Se and Cd. For Hg analysis, the samples were prepared and extracted using methods described by Monk [22]; determinations were made using the method of Hatch and Ott [23]. Analysis for Fl was conducted using the method outlined by Singer and Armstrong [24], with some modifications [25,26]. The analytical methods for the other elements followed recommended procedures [27]. The concentrations of all elements were based on wet weight, except those of Fl in femurs, which were based on dry weight. Concentrations were not corrected for recovery values from spiked samples. Mean moisture levels were 74% in the livers and 78% in the kidneys of trumpeter swans.

RESULTS

Relationship of Pb concentration to swan mortality

The final diagnoses and Pb concentrations in livers are presented in Tables 1 and 2 for 72 trumpeter swans found dead in seven western states from 1976 to 1987. Of the 67 swan livers analyzed for Pb, 51 (76%) contained detectable concentrations; 18 contained levels of Pb generally considered lethal or else some of the characteristic lesions of lead poisoning found in waterfowl [1,2]. The latter group included two male subadult swans with relatively low amounts of Pb (3.6 and 4.9 μg/g) that were diagnosed as dying from lead poisoning. Both of the subadults with low Pb levels died during 1985; each was extremely emaciated and exhibited hyperkeratosis (sloughing) of the gizzard lining, partial impaction of the digestive tract and an enlarged gall bladder. Only one exhibited a bile-stained gizzard lining. The maximum amount of Pb in the liver of a swan was 37 μg/g. Pb concentrations in livers that are associated with waterfowl mortality in experimental studies usually range from 20 to 40 μg/g [28], but lethal levels extend from 5 μg/g [29] to 80 μg/g [30]. Wild mute swans apparently died from lead poisoning with as little as 10 to 14 μg/g dry weight (approximately 3 to 4 μg/g wet weight) in their livers [6,11].

The mortality of swans from lead poisoning varied widely from area to area; the highest incidence was 62% (8 of 13) of those found dead in Washington, whereas none of the 5 swans from Alaska and Wyoming died from lead poisoning (Tables 1 and 2). In the tri-state population of Idaho, Montana and Wyoming, 20% of the swan mortality was induced by lead toxicosis. We examined the gizzard contents of the 18 trumpeter swans that died from lead poisoning and found ingested lead artifacts in 13 (72%); 9 contained shot and 4 contained fishing sinkers (Table 1). A single ingested lead shotgun pellet was detected in the gizzard contents of each of two swans with low Pb levels (0.60 and 1.3 μg/g) in their livers.

Most dead swans were obtained in the tri-state area (Table 2); these included local birds and wintering birds from other areas such as Alaska. The recent increase in the number of diagnosed swan mortalities probably reflects a greater frequency of reporting and submitting specimens for examination rather than an increase in the number of mortalities. Most of the mortalities involved one or a few birds at a time, but a die-off of about 50 swans occurred on the Henry's Fork of the Snake

River, Idaho, in March and April 1985. Most of these deaths appeared to be related to the icing over of traditional winter feeding sites as a result of reduced stream flow (Justin Naterman, personal communication); however, five swans died from lead poisoning (Table 1).

Suspected lead-induced mortality in trumpeter swans (Table 3) has occurred in British Columbia since at least 1925 (17,18,20,31,32). There also are several areas in western Washington where relatively large numbers of trumpeter and tundra swans die from lead poisoning; these include the Willapa National Wildlife Refuge (WNWR) near the Pacific Ocean and several sites in Skagit County, north of Seattle. A summary of necropsy and Pb analyses data for 84 wintering trumpeter swans found dead in Skagit County from 1976–77 through 1985–86 (Mike Davison, personal communication) revealed that lead poisoning accounted for 23 (27%) of the deaths. During the winter of 1986–87, five trumpeter swans died from lead toxicosis in Skagit County and several died in nearby British Columbia; the gizzards of birds from Skagit county contained from 3 to 20 ingested lead shot (Martha Jordan, personal communication). Two cygnets from RRLNWR in Montana were suspected of dying from lead poisoning (Table 3); each contained ingested lead shot [23].

Concentrations of Pb in blood

Blood was sampled in 35 trumpeter swans in 1984 and 1985 (Table 4); all of the swans seemed healthy, except the moribund bird found at Harriman State Park (HSP) in Idaho in March 1985. Lead was detected in the blood of 16 of 25 swans from RRLNWR, 0 of 8 swans from MNWR, and in both swans from Wyoming and HSP (Table 4). Generally, the Pb level was low in most blood samples, including the sick swan from HSP, but 0.71 μg/g Pb was detected in the blood of a breeding female from RRLNWR. The blood samples from MNWR were taken in cygnets captured in August and September. The blood samples from 10 trumpeter swans collected in 1985 in Wyoming contained from 0.07 to 0.68 μg/g, with a geometric mean of 0.16 μg/g (Ruth Gale, personal communication). These values are similar to those for the blood of swans from RRLNWR (Table 4).

Concentrations of nonplumbous elements

Concentrations of 13 other elements in the livers did not appear elevated, except that the concentration of Fe was very high (3,800 μg/g) in a trumpeter swan that died of lead poisoning

Table 1. Lead concentrations, lead poisoning and other factors related to mortality of trumpeter swans, 1976 to 1987

Area where found dead[a]	Date	Sex	Age[b]	Liver Pb[c] (μg/g, wet wt.)	No. of ingested lead artifacts in gizzard: Shotgun pellets	Fishing sinkers	Diagnosis
Washington							
WNWR	16 Jan. 1979	M	Adult	10.4		5	Pb poisoning
WNWR	4 Feb. 1979	M	Adult	7.5	17		Pb poisoning
WNWR	20 Feb. 1980	Unknown	Unknown	21.8	3		Pb poisoning
WNWR	19 Dec. 1984	Unknown	Subadult	37.0	28		Pb poisoning
PSSM	5 Feb. 1976	M	Adult	ND			Aspergillosis
PSSM	5 Feb. 1976	F	Subadult	0.9			Open[d]
PSSM	5 Feb. 1976	M	Subadult	ND			Gunshot
PSSM	5 Feb. 1976	F	Adult	ND			Aspergillosis
LTWMA	1983	M	Adult	20.0	1		Pb poisoning
LTWMA	1983	F	Adult	17.1			Pb poisoning
BL[e]	Jan.–Feb. 1981	M	Adult	21.9	7		Pb poisoning
BL[e]	Jan.–Feb. 1981	F	Adult	1.3	1		Open

continued

Table 1 continued.

Area where found dead[a]	Date	Sex	Age[b]	Liver Pb[c] (μg/g, wet wt.)	No. of ingested lead artifacts in gizzard: Shotgun pellets	No. of ingested lead artifacts in gizzard: Fishing sinkers	Diagnosis
Washington (continued)							
BL[e]	Jan.-Feb. 1981	F	Adult	19.7	68		Pb poisoning
TNWR	8 Nov. 1982	F	Adult	NA			Gunshot
Alaska							
KNWR	9 Sept. 1983	M	Cygnet	4.2			Fibrinous peritonitis, anemia, intestinal parasitism, possible predation
KNWR	16 Sept. 1983	F	Cygnet	3.4			Open (anemia, possible predation)
KNWR	7 Nov. 1983	Unknown	Unknown	NA			Open
KNWR	19 Nov. 1983	Unknown	Unknown	0.43			Open (anemia, possible predation)
Nevada							
RLNWR	15 Mar. 1984	F	Subadult	16.7		1	Pb poisoning
RLNWR	6 Mar. 1984	M	Subadult	0.58			Open (emaciation)
Tri-state area (Idaho, Montana, Wyoming)							
RRLNWR	18 Aug. 1977	F	Cygnet	ND			Pneumonitis, fractured liver
RRLNWR	18 Aug. 1977	M	Cygnet	ND			Open (pneumonitis)
RRLNWR	16 Aug. 1983	F	Adult	0.20			Impaction of colon and cloaca, severe necrotizing colitis
RRLNWR	12 Sept. 1983	F	Subadult	ND			Trauma, broken neck
RRLNWR	24 Jan. 1984	F	Unknown	20.0	5		Pb poisoning
RRLNWR	21 May 1984	M	Unknown	0.68			Chronic peritonitis and pericarditis
RRLNWR	14 June 1984	M	Unknown	ND			Open
RRLNWR	28 Feb. 1985	F	Adult	0.44			Avian tuberculosis, granulomatous pneumonia, pododermatitis, aspergillosis
RRLNWR	28 Feb. 1985	M	Subadult	4.9			Possible Pb poisoning
RRLNWR	1 Aug. 1985	F	Adult	0.05			Open (emaciation)
RRLNWR	2 Jan. 1986	Unknown	Unknown	0.21			Open (possible predation)
RRLNWR	23 May 1986	F	Adult	0.16			Fractured wing, parasitism, emaciation (euthanized)
RRLNWR	11 June 1986	F	Adult	0.23			Open
RRLNWR	19 Oct. 1986	F	Adult	ND			Gunshot
RRLNWR	1 Dec. 1986	M	Subadult	0.60	1		Renal coccidiosis, dehydration, kidney failure
RRLNWR	5 Dec. 1986	F	Subadult	11.3	59		Pb poisoning
YNP	27 Feb. 1980	F	Subadult	0.18			Esophageal abscess (embedded fishhook)
TNF	9 July 1980	Unknown	Cygnet	0.40			Open
JAWY	1 Dec. 1980	F	Adult	NA			Trauma (impact with power pole)
JAWY	6 Feb. 1985	F	Subadult	ND			Trauma (apparent impact with fence)
HFID	Apr. 1979	F	Adult	ND			Open
HFID	Apr. 1979	F	Subadult	ND			Open

HFID	Apr. 1979	F	Subadult	ND			Emaciation, intestinal blockage
HFID	29 Jan. 1980	F	Subadult	ND			Open
HFID	28 Nov. 1980	F	Unknown	NA			Peritonitis, impaction
HFID	17 Apr. 1984	F	Unknown	0.49			Gunshot
HFID	10 Mar. 1985	F	Adult	0.43			Myocarditis (*Sarconema* sp.), coccidiosis (*Eimeria brontae*), nasal leeches (*Theromyzon* sp.)
HFID	9 Apr. 1985	F	Adult	21.0		1	Pb poisoning, myocarditis (*Sarconema* sp.)
HFID	9 Apr. 1985	M	Subadult	0.47			Emaciation, esophagitis, drowning, renal coccidiosis, visceral gout
HFID	9 Apr. 1985	F	Subadult	0.43			Esophagitis (*Echinuria* sp.), impaction, emaciation
HFID	9 Apr. 1985	F	Adult	21.0		1	Pb poisoning
HFID	9 Apr. 1985	F	Subadult	1.5			Coccidiosis (*Eimeria* sp.), esophagitis (*Echinuria* sp.)
HFID	9 Apr. 1985	F	Adult	0.45			Coccidiosis (*Eimeria* sp.)
HFID	9 Apr. 1985	M	Subadult	1.1			Emaciation, myocarditis (*Sarconema* sp.)
HFID	9 Apr. 1985	M	Subadult	2.4			Emaciation, coccidiosis
HFID	9 Apr. 1985	M	Subadult	3.6			Possible Pb poisoning
HFID	9 Apr. 1985	M	Unknown	7.7			Pb poisoning, myocarditis (*Sarconema* sp.), parasitic enteritis
HFID	9 Apr. 1985	M	Unknown	0.40			Emaciation, sinusitis (nasal leeches, *Theromyzon* sp.)
HFID	9 Apr. 1985	F	Adult	9.4			Pb poisoning
HFID	9 Apr. 1985	Unknown	Subadult	0.26			Coccidiosis (*Eimeria* sp.)
HFID	9 Apr. 1985	M	Adult	12.0	3		Pb poisoning
HFID	15 Apr. 1985	M	Adult	0.44			Pericarditis, valvular endocarditis
HFID	15 Apr. 1985	F	Adult	1.8			Open
HFID	15 Apr. 1985	M	Unknown	0.45			Open
HFID	Dec. 1985	M	Subadult	0.02			Parasitism, nasal leeches (*Theromyzon* sp.), emaciation
HFID	15 Jan. 1986	M	Subadult	0.02			Open (emaciation)
HFID	6 Mar. 1986	M	Subadult	0.78			Intestinal tapeworm (*Sobolevicanthus* sp.) parasitism, proventriculitis, emaciation
Oregon							
MNWR	16 Aug. 1984	M	Cygnet	NA			Myocarditis
MNWR	5 Jan. 1985	F	Subadult	ND			Gunshot
MNWR	5 Jan. 1985	F	Subadult	ND			Gunshot
WV	6 Mar. 1986	M	Subadult	0.38			Gunshot
MNWR	11 Mar. 1987	M	Subadult	ND			Open

[a]WNWR, Willapa National Wildlife Refuge; PSSM, Puget Sound–Skagit Marshes; LTWMA, Lake Terrell Wildlife Management Area near Bellingham; BL, Barney Lake near Mount Vernon; TNWR, Turnbull National Wildlife Refuge near Spokane; KNWR, Kenai National Wildlife Refuge; RLNWR, Ruby Lake National Wildlife Refuge; RRLNWR, Red Rock Lakes National Wildlife Refuge and nearby area in the Centennial Valley, Montana; YNP, Yellowstone National Park; TNF, Targhee National Forest; JAWY, Jackson, Wyoming; HFID, Henry's Fork of the Snake River and adjacent areas of Idaho, including Harriman State Park; MNWR, Malheur National Wildlife Refuge; WV, Willamette Valley near Airlie.

[b]A subadult is a nonbreeding bird that has not attained adult plumage; the cygnet category also includes young of the year.

[c]NA, liver not analyzed; ND, no Pb detected. 4 of 5 femurs of swans from RRLNWR and HFID contained Pb (range = ND to 11 $\mu g/g$); the swan with highest femur concentration died from lead poisoning.

[d]Necropsy failed to reveal probable cause of death.

[e]Data from Tener [18].

Table 2. Origin of trumpeter swan specimens and probable mortality from lead poisoning

State	No. of swans found dead	No. of livers: Analyzed for Pb	No. of livers: With probable lethal levels[a]	% mortality from lead poisoning
Alaska	4	3	0	0.0
Idaho	28	27	6	22.2
Montana	16	16	3	18.8
Nevada	2	2	1	50.0
Oregon	5	4	0	0.0
Washington	14	13	8	61.5
Wyoming	3	2	0	0.0
Totals	72	67	18	25.0

[a]Pb levels of >3.6 μg/g in combination with several signs of lead toxicosis.

(Table 5); elevated Fe levels are typically found in livers of lead-poisoned birds [2]. There was a wide range of Cu levels in the livers (4.8 to 240 μg/g), but the highest concentrations of this essential element and of other elements in liver samples and of Cd in kidney samples were substantially below those associated with mortality and reproductive problems in birds [33]. The Fl levels in the femurs of two swans were within the range detected in femurs of experimental eastern screech-owls (*Otus asio*) that experienced some reproductive problems; however, the extremely variable screech-owl data merely indicated exposure and were of little value in assessing hazard [26].

With few exceptions, the concentrations of other metals and trace elements in blood were not unusually high; Ni, As and Cd were not detected in the samples from RRLNWR and MNWR (Ta-

Table 3. Records of suspected[a] lead-induced mortality of trumpeter swans, 1925 to 1983

Area where found dead[b]	Date	No. of swans: Total	No. of swans: Containing ingested lead shot in gizzard (%)	No. of lead shot per bird	Ref.
RRLNWR	1937	4	4 (100)	3–19	[35]
VLBC	1925	9	Several	≤451	[17]
VIBC	1946	13	13 (100)	2–29	[20]
RRLNWR	1960	1[c]	1 (100)	1	–[d]
RRLNWR	1963	1[e]	1 (100)	115	–[d]
VIBC	1945	3	1 (33)	30	[31,32]
RRLNWR	1983	1	1 (100)	32	–[d]

[a]No analyses for Pb; some birds were not necropsied.
[b]RRLNWR, Red Rock Lakes National Wildlife Refuge, Montana; VLBC, Vaseaux Lake, British Columbia; VIBC, Vancouver Island, British Columbia.
[c]3-week-old cygnet.
[d]Ruth Gale, personal communication.
[e]Cygnet.

Table 4. Concentrations (μg/g, wet wt.) of Pb in blood of trumpeter swans, 1984 to 1985

Area[a]	Date	N	No. of positive samples	Pb concentrations: Geometric mean	Pb concentrations: 95% confidence limits	Pb concentrations: Range[b]
RRLNWR	July 1984 and 1985	25[c]	16	0.09	0.06–0.15	ND–0.71
MNWR	Aug.–Sept. 1984	8[d]	0	–	–	ND
JAWY	July 1984	1[e]	1	–	–	0.28
HSP	Mar. 1985	1[f]	1	–	–	0.16

[a]See Table 1 for abbreviations; HSP, Harriman State Park.
[b]ND, not detected.
[c]Includes adults and subadults.
[d]All cygnets.
[e]Female adult.
[f]Sick bird, age and sex not available.

Table 5. Nonplumbous elements (μg/g, wet wt.) in liver, kidney, femur and blood of trumpeter swans, 1983 to 1985

	Cu	Zn	Ni	Mg	Fe	Hg	Mn	As	Se	Cd	Fl
Liver, kidney and femur (tri-state population)[a]											
N (Positive N)[b]	13 (13)	4 (4)	4 (1)	4 (4)	4 (4)	4 (4)	4 (4)	1 (0)	4 (4)	3 (3)	4 (4)
GM	33	96		201	668	0.10	2.1		1.2	1.6	1,185
Range	4.8–240	61–160	ND–0.10	176–260	54–3,800	0.03–0.63	1.1–3.4	ND	0.8–1.7	0.37–4.0	860–1,700
Blood (tri-state population)											
N (Positive N)	10 (5)	10 (10)	8 (0)			9 (2)		9 (0)	12 (8)	14 (2)	
GM	0.09	4.7							0.17		
Range	ND–0.69	3.7–8.8	ND			ND–0.09		ND	ND–2.95	ND–0.13	
Blood (MNWR)											
N (Positive N)	3 (3)	3 (3)	8 (0)			3 (0)		3 (0)	3 (0)	4 (0)	
GM	0.21	5.4									
Range	0.14–0.30	5.2–5.7	ND			ND		ND	ND	ND	

[a]Analyzed for Cd in kidney, Fl in femur, and all other elements in liver including Al (2 of 4 samples positive; GM = 1.1 μg/g, range = ND–3.1 μg/g) and Cr (4 of 4 samples positive; GM = 0.20 μg/g, range = 0.09–0.36 μg/g).
[b]N, total samples analyzed; positie N, number of samples analyzed that contained the element; GM, geometric mean; ND, no concentration detected; a blank indicates calculation not possible or no analysis.

ble 5). Data useful for interpreting the concentrations of some elements in blood and other tissues are generally unavailable. The highest Se concentration of 2.95 μg/g in blood is apparently elevated, but it is much lower than levels associated with problems in experimental birds (Gary Heinz, personal communication). The concentrations of metals and trace elements in the blood of cygnets from MNWR were especially low.

DISCUSSION

Trumpeter swans seem particularly vulnerable to lead-induced toxicosis associated with ingestion of shot and fishing sinkers because of their method of feeding and a seemingly high susceptibility to lead toxicosis. The typical feeding method of trumpeter swans involves digging up large amounts of bottom sediments of lakes and streams [34]. The large volume of plant material and sediments ingested increases the likelihood of the birds' ingesting lead shot or sinkers—such as the 451 shot recovered from the gizzard of a swan in British Columbia [17], which was surpassed only by the 944 shot in the gizzard of a mute swan in Scotland [11]. Also, swans in general appear unusually sensitive to lead toxicosis because severe pathological changes were associated with particularly low Pb levels in some of our trumpeter swans and in mute swans from another study [6]. Regardless of inherent sensitivity, lead poisoning in swans is influenced by a number of factors including weather, water levels, soil, shot availability and diet [13].

Cygnets as well as older swans are at risk from ingestion of lead shot; a three-week-old trumpeter swan found dead at RRLNWR contained a single shot in its gizzard and a second cygnet found dead there in October had ingested 115 shot (Ruth Gale, personal communication). Lead poisoning due to fishing sinkers was apparently first described in mute swans in England in the late 1970s [3,5]; some cygnets ingest sufficient lead from such sources to elevate blood levels to 37 μg/g [35]. Ten cygnets died from lead poisoning on the Thames River in 1980 and 1981; the ages of these birds ranged from 10 to 20 weeks [6].

Although there has been waterfowl hunting at RRLNWR since its establishment and lead toxicosis from ingested shot was documented in trumpeter swans as early as 1937 [34], lead poisoning was not considered a major factor in the recent population decline [36]. Analysis of banding and resighting data from 1949 to 1982 for trumpeter swans in the RRLNWR and nearby areas revealed a relatively high survival rate that did not decrease as the population declined; unfortunately, the data were considered inadequate to assess changes in survival rates [37]. Our data indicate that about 20% of the known trumpeter swan mortality in the RRLNWR and nearby areas occupied by the tri-state population is related to lead toxicosis from ingested shot and fishing sinkers. The conspicuous size and white color of the trumpeter swan ensures that a relatively large percentage of dead birds will be observed. In recent years, there also has been an apparent increase in the number of swans reported, necropsied and analyzed for contaminants.

The incidence of mortality from lead artifacts is much lower than that reported for mute swans in England, which experienced a population decline associated with excessive mortality from ingestion of lead fishing sinkers. In affected segments of the population, 43 to 76% of known mortality was related to lead poisoning [4]. This seems the best evidence of lead artifacts' depressing a population of birds. Certain "hot spots" exist in western Washington and British Columbia where relatively large numbers of trumpeter swans die from lead toxicosis induced by shot ingestion, but these include many wintering birds from the large population in Alaska. In the tri-state area, where the breeding population is declining and productivity has been low for the last several decades, the role of lead-induced mortality in the population decline seems uncertain. The 20% of known mortality in permanent and winter residents attributable to lead artifacts is much lower than that found in the declining mute swan population in England and in localized areas of the United States where up to 84% of the ducks and 73% of the geese found dead were victims of lead poisoning [2]. The percentage of known mortality induced by lead artifacts is merely an indicator and may or may not be directly proportional to the impact of lead on the population as a whole.

There is no evidence that lead is directly affecting the reproductive success of swans in the tri-state population. The abnormally developed feet of a few cygnets reported in earlier studies [21,34] are more characteristic of defects related to nutritional deficiencies than of those induced by contaminants. The amounts of lead detected in the blood of seemingly healthy swans from RRLNWR and Wyoming in 1984 and 1985 were relatively low and were similar to those in a healthy population of mute swans in Denmark [38]. Mute swans exposed to fishing sinkers in England had maximum amounts of Pb in the blood of 37 $\mu g/g$, and one bird survived with 6.8 $\mu g/g$ [35]. Pb levels in the blood of most of these swans exceeded the maximum acceptable level of 0.4 $\mu g/g$ established in studies of these swans in England; adverse effects on reproduction evidently occurred when levels exceeded 2 $\mu g/g$ [7].

The recent inclusion of RRLNWR in a "steel shot zone," where hunting with shells containing lead shot is prohibited, and the decreased use of lead shot in other waterfowl hunting areas will probably decrease the risk of lead poisoning to swans and other birds [39]. Efforts to replace lead fishing weights with nontoxic materials are under way in England [5,6]; this contingency should be considered for problem areas in North America such as the Henry's Fork of the Snake River.

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